THE CLINICAL RECOGNITION OF CORONARY HEART DISEASE*

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Over ninety per cent of the lesions which affect the coronary arteries are due to atherosclerosis and it is with this group that the present discussion is concerned. Coronary heart disease occupies a dominant place in clinical medicine because of the large number of individuals involved, because it kills or impairs the efficiency of many who are in the most productive period of life and because the pictures which it presents are so varied in their manifestations. It offers a challenge in that the fundamental mechanisms involved in the production of arterial degeneration are still imperfectly understood.

HISTORICAL BACKGROUND

The earliest observations correlating advanced disease of the coronary arteries with serious illness were probably those of Théophile Bonet of Geneva.[1] In 1700, he described the case of a middle aged poet who died shortly after a paroxysm of dyspnea which may well have been an anginal seizure. At autopsy there was found ossification of the coronary arteries and these were almost completely occluded.

There are two prominent landmarks in the development of our knowledge. The first is the description of angina pectoris by William Heberden in 1768.[2] Heberden, however, was not aware that the chest pangs which he described were connected with the coronary arteries. It was Jenner who pointed out this relationship several years later. The second is the account of coronary thrombosis by James B. Herrick as given in his papers published in 1912 and 1919.[3, 4] Herrick's story of his efforts to acquaint the medical profession with the clinical features of coronary heart disease is told in his recent autobiography which he

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has called "Memories of Eighty Years." [5] It is characteristic of the reluctance with which new viewpoints so often are received.

"In 1912," he says, "my paper on 'Clinical Features of Sudden Obstruction of the Coronary Arteries' appeared, based on a case of coronary thrombosis in which I had made an ante mortem diagnosis. Recognizing the radical nature of the views I held, which led me to conclude that this condition was not, as was then the belief, merely a pathologic curiosity but in reality a clinical entity with symptoms that often made it possible to diagnose it during life, I postponed publication for some time until, by search of the literature and further observation, I had reached conclusions that seemed to me justifiable and sound. The paper when read in 1912 before the Association of American Physicians aroused no interest. It fell like a dud." It was seven years later, after the publication of his second paper, to quote further, that "physicians in America and later in Europe woke up, and coronary thrombosis came into its own, to become later a household word translated by the layman into 'heart attack.' "

It has always seemed to me that the contribution of Ernst von Leyden of Danzig, published in 1884,[6] has not been given the recognition which it deserves. Leyden was a good pathologist and an excellent clinician. He divided coronary sclerosis, with respect to its effects, into four groups: (1) sclerosis without disturbance of heart function, that is without symptoms; (2) acute thrombotic softening or hemorrhagic infarction; (3) a chronic form leading to disseminated or diffuse fibrous degeneration of the myocardium and, in some instances, to ventricular aneurysm; (4) a combined form in which is found an old fibrotic process as well as acute thrombotic softening. He also described the group of acute cases with sudden death. It is surprising that this approach, which resembles so closely our current views, was relatively ignored even in Germany where these and other pertinent observations had been made.

Recognition in a Modern Hospital

Eighteen years ago an attempt was made to trace the development of awareness of the coronary problem in the wards and autopsy room at the Presbyterian Hospital in New York by charting and comparing the incidence of clinical and pathologic diagnoses during the period from 1910 to 1931.[7] These observations, published in 1934, have now been

TABLE I.—CLINICAL AND AUTOPSY INCIDENCE OF CORONARY DIAGNOSES AT FIVE-YEAR INTERVALS, COLUMBIA-PRESBYTERIAN MEDICAL CENTER, 1910-1950*

Year	*Total Hospital Admissions*	*Clinical Coronary Diagnoses*	*Total Autopsies*	*Autopsy Coronary Diagnoses*	*Rate of Clinical Coronary Diagnoses per 1000 Admissions*	*Per Cent of Total Autopsies with Coronary Diagnoses*
1910	4,038	.....	111	11	.....	9.9
1915	3,843	2	113	21	0.5	18.6
1920	4,300	11	126	43	2.6	34.1
1925	4,092	19	117	44	4.6	37.6
1930	14,788	107	231	68	7.2	29.4
1935	17,134	132	304	92	7.7	30.3
1940	19,878	173	292	111	8.7	38.0
1945	21,427	213	283	112	9.9	39.6
1950	25,474	228	295	141	9.0	47.8

* The composition of the hospital population varied somewhat during the period covered. Ward, semi-private and private patients are included. From 1930 on, Sloane Hospital (with its newborn) is included. From 1940 on, Eye Institute is included, but Babies Hospital, Neurological Institute, Orthopedic Hospital and Mary Harkness Convalescent Home are excluded.

extended to cover the period from 1910 to 1950, and since 1930, cases in various affiliated institutions in the Columbia-Presbyterian Medical Center have been included. The figures obtained during a year have been tabulated at 5-year intervals. In this way certain facts and trends have become apparent (Table I).

It is clear that in these data are inherent certain errors which cannot be avoided. There is variation in the character of the hospital population with respect to disease, age and sex. There is variation also in the composition of the autopsy material with respect to the types of diseases observed. The slight changes in the figures of Table I, due to the modification of the hospital population by the later inclusion or exclusion of certain of the affiliated institutions caring for special types of cases, may well have been due to chance variations. For estimating the frequency with which the clinical diagnosis of coronary disease was made only those records were used in which there was specific mention of coronary involvement. In many instances it seems certain that, in the absence of manifest symptoms or signs, or because interest in some other condition was dominant, this diagnosis was not recorded in the final appraisal of the case. Such circumstances obviously would lower

the figures for incidence. In the autopsy group all types and degrees of coronary lesions were included. As was indicated in our previous publication, more than 97 per cent were arteriosclerotic.

The results, shown in the final two columns of Table I, have been reviewed to determine whether some of the fluctuations might be merely chance variations.* It is noteworthy that the term coronary disease does not appear as a final diagnosis in any history in 1910. In going over some of the old files, one finds reference to angina pectoris but none to coronary thrombosis or myocardial infarction. Such cases were designated as chronic myocarditis, cardiac insufficiency or, occasionally, pyrexia of unknown origin. This latter group was shown at autopsy to represent instances of cardiac infarction with fever. The general trend in frequency over the entire period shows a statistically significant increase. The relationship of sudden increases to the publication of Herrick's observations is of considerable interest. In 1912 his first paper appeared. Although generally neglected, his observations made some impression at the Presbyterian Hospital because the diagnosis of coronary disease was made twice during the year 1915. In 1919, after publication of his second paper, there was a larger upward jump in the rate of clinical diagnosis from 0.5 to 2.5 per thousand admissions. From then on, there occurred a gradually progressive increase in the number of coronary diagnoses, although the change from year to year was not statistically significant. However, the difference between the figure of 4.6 in 1925 and of 9.0 in 1950 is significant. During the past two decades there has been no important increase.

The pathologists recognized and recorded coronary diagnoses even in 1910. Again the trend during the next 40 years is statistically significant. As was the case among the clinicians, the pathologists were made more aware of the importance of the coronary arteries in relation to the clinical features of disease after Herrick's first paper and there is a rise from 9.9 per cent in 1910 to 18.6 in 1915. Again, after Herrick's second paper, there is a significant increase from 18.6 to 34.1. From then on, the variations are only of borderline significance or not significant at all.

In short, during the past 40 years the number of clinical diagnoses of coronary heart disease has increased in frequency in a large university medical center. Greater diagnostic skill probably was largely responsible,

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particularly in the earlier years. The diminution in the number of patients with acute infectious diseases and the aging of the population, as well as other changes in the character of those admitted to the hospital, are factors which must also be considered. Although prior to 1920 there is a trend upward of the incidence found at autopsy, the slope of the curve is not as sharp as that depicting the frequency of clinical diagnoses. In fact, in the later years, the incidence appears to be essentially constant. Within the limitations of the character of the autopsy material, it seems that the clinicians, with the aid of more accurate methods of diagnosis, are now recognizing conditions during life which have for a much longer period been apparent to the pathologists.

No claim is made that these figures are generally applicable and represent an over-all picture. They are, however, probably more reliable than any which could be obtained from death certificates. A comparable study was recently published by Morris,[8] who analyzed necropsy records of the London Hospital where the coronary arteries, as well as the myocardium, have been routinely examined since 1907. Some 6000 records from this hospital were studied from that date to 1949. They showed a 7-fold increase during this period in the number of cases of coronary heart disease. Morris concluded that coronary sclerosis and myocardial infarction, rare before the first World War, have since become common and that this increase represents a rising incidence rather than better recognition. Our own observations suggest that both factors probably are concerned but tend to place the preponderant emphasis on greater accuracy in diagnosis.

Clinical Recognition

The diagnostic approach to any group of diseases requires a logical classification for proper understanding. Atherosclerosis of the coronary arteries causes a variety of pathologic lesions, both in the vessels and in the heart muscle. From the point of view of the physician who treats the patient, it is the resulting disturbances in function which are the chief concern. The one common denominator at the basis of the manifestations of all coronary disease is coronary insufficiency. This may be defined as the disorder in which the amount or quality of blood is inadequate to meet the demands of the myocardium if it is to function as an efficient pumping mechanism. Oxygen is essential for effective muscular contraction; hypoxia or anoxia of the myocardium is the fundamental

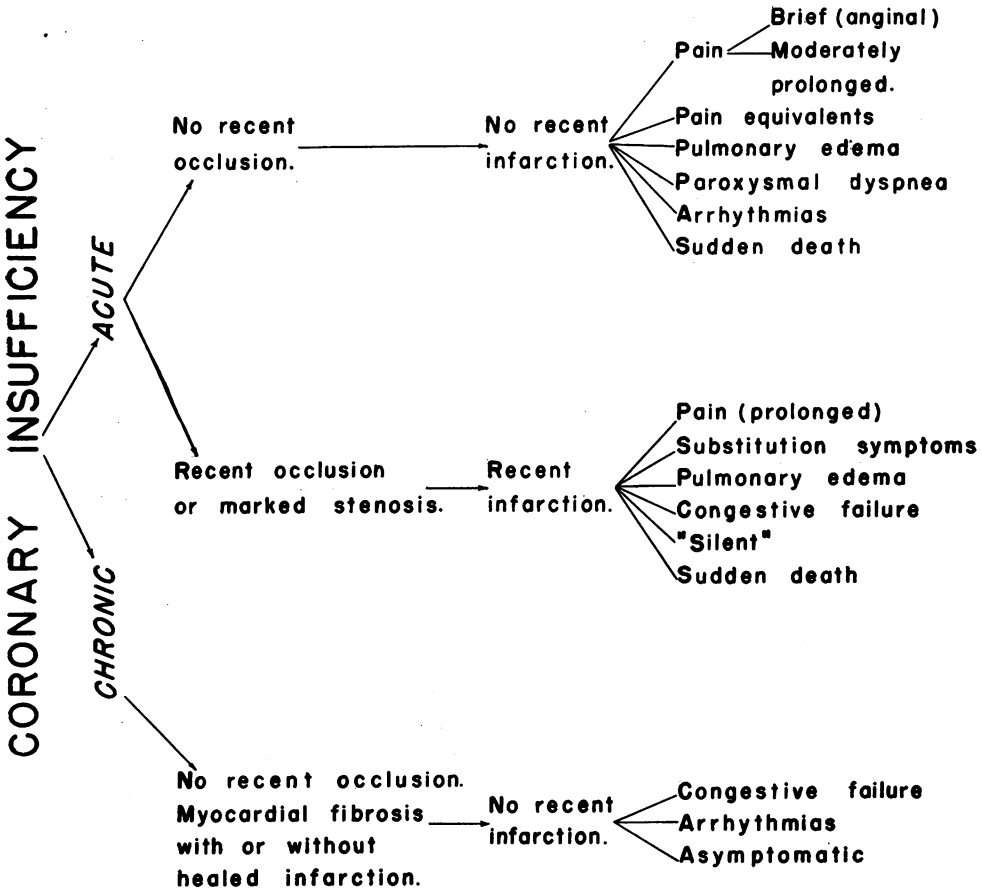

Fig. 1—Clinical features of coronary insufficiency.

cause for the disturbances produced. These may result from various anatomic or physiologic lesions.

In Figure 1, there is presented a schema which, it is believed, includes the various clinical features of coronary heart disease on the basis of coronary insufficiency. It offers a simple concept and one in which there is unity of cause.

Coronary insufficiency may be acute or chronic. In the *acute form*, there may be no recent occlusion and no recent infarction. In this variety pain is the most common symptom. Discomfort may be brief, of the anginal variety, or it may be more prolonged, lasting 20 minutes or more and yet not followed by cardiac infarction. This is the type of

pain intermediate between the discomfort of an anginal attack and that associated with occlusion and infarction. It has been designated by Blumgart[9] as "coronary failure"; but merely because of its duration, it does not merit a name of its own. There are certain pain equivalents, such as weakness, sweating or a sense of pressure or tightness across the chest. Discomfort may be referred to the abdomen and regarded as a digestive disorder. At times pulmonary edema or paroxysmal dyspnea will usher in an attack. Focal anoxia may initiate an arrhythmia. Sudden death in the presence of an impaired coronary circulation may follow some unusual exertion or emotional experience; it is to this type that the term "acute, fatal coronary insufficiency" has been applied.[10]

Recent occlusion of a vessel, whether by a thrombus or atheroma, or marked stenosis may cause fresh infarction. Under these conditions, pain is apt to be more prolonged and not infrequently is associated with nausea or vomiting. Again there may be substitution symptoms, such as mild substernal pressure, general weakness, sweating about the head and neck or digestive disturbances. Pulmonary edema sometimes is observed following sudden closure and congestive failure may follow when the collateral circulation is inadequate to nourish the remaining uninjured heart muscle. Occlusion may be without discomfort and sometimes occurs "silently," particularly in the old and debilitated. Sudden closure may initiate ventricular fibrillation and be followed shortly by sudden death.

In the *chronic form* there is no recent occlusion and no recent infarction. The common finding is myocardial fibrosis with or without healed infarction. This is the condition to which formerly was applied the term "chronic myocarditis." The heart is usually enlarged and congestive failure is frequent. There may be arrhythmias of various sorts, including premature contractions, auricular fibrillation or flutter, or varying degrees of auriculoventricular heart block. Sometimes the condition is asymptomatic and is discovered only when the electrocardiogram shows bundle branch block, significant T-wave negativity or other evidence of old myocardial injury.

From the point of view of the attending physician, the most important diagnostic problem is to determine whether recent infarction has taken place, for upon its presence or absence will depend the general management of the patient. The criteria for the diagnosis of infarction are now well established; but it is proper to emphasize again that the

picture of coronary heart disease is varied and that its recognition sometimes presents real difficulties. There is no classic pattern.

Diagnostic Aids

Sclerosis without recent occlusion. There is no form of examination which is as important as the taking of a good history. This has been said many times but still is not sufficiently appreciated. The physician who is responsible for the care of the patient should be the one to hear from the sufferer's own lips his history of discomfort. In many cases such an account, if skillfully obtained, suffices for diagnosis; it may be the only evidence obtainable, for the physical examination and all special tests may prove to be negative.

On physical examination, the presence of cardiac enlargement, in the absence of hypertension, valvular disease or other manifest cause, should always arouse suspicion. This should be confirmed by x-ray or orthodiagraphic examination. Changes in the form of the electrocardiogram may afford graphic evidence of myocardial injury. Of particular importance are bundle branch block, significant T-wave negativity and arrhythmias, particularly auriculoventricular block or auricular fibrillation or flutter. It should always be kept in mind that alterations in the electrocardiogram are not etiologically specific and that they must be interpreted in terms of the clinical picture as a whole; it is not possible to make a diagnosis of coronary heart disease on the basis of the electrocardiogram alone.

In recent years, the ballistocardiograph has become of increasing interest as a diagnostic tool. In its original forms, whether damped or undamped, it is a cumbersome and expensive instrument requiring considerable technical skill for its proper manipulation. The correct interpretation of ballistocardiographic records likewise demands experience and special knowledge. In the hands of Starr and others such tracings have afforded early evidence of myocardial dysfunction and have suggested coronary insufficiency at a time when other methods failed to indicate its presence.[11] Results obtained with the use of smaller instruments, such as that devised by Dock,[12] should, for the present, be accepted with caution. These devices are more susceptible to oscillations produced outside the body than are the table models. Whether records obtained with the smaller machines are wholly reliable is still open to question. It seems likely that a relatively simple and accurate ballisto-

cardiograph will be developed in the near future. The practical usefulness of such an apparatus is still to be determined.

In a small group of cases, the history and examination leave the physician in doubt as to whether he is dealing with a coronary problem. Under these circumstances, two special stress tests have been devised. The two-step exercise test of Master[13] is comparatively simple to perform. The chief objections to it are that (1) criteria of a positive response are not well defined and (2) false positives occur.[14] These, in our opinion, are serious faults. Furthermore, we are not convinced of the claim made by the sponsors of this test that a negative result excludes coronary insufficiency.

Our own studies have been concerned more particularly with the anoxemia test, during which the patient is made to breathe, at the normal rate of respiration, a mixture of 10 per cent oxygen and 90 per cent nitrogen.[15-17] This test has now been used extensively both in this country and abroad and, with the exercise of the simple precautions which have been outlined, is entirely safe. Anoxia is quickly reversible because 100 per cent oxygen can quickly be given. It should be used only in cases in which the diagnosis is in doubt. The criteria for a positive effect have been clearly stated and in thousands of tests performed in many clinics have been found to be valid. A recent report by Nylin,[18] of Stockholm, presents favorable results in 1130 tests. In the light of a large experience, we can reiterate the statement that a positive test is a sign of coronary insufficiency; on the other hand, a negative test does not rule out the possibility that lesser degrees of coronary insufficiency may be present.

The following case report relates an instance in which the test was a decisive factor in diagnosis.

C. G. (Unit No. 842455) was a jewelry salesman, 58 years of age, and was referred from another hospital because of pain in the chest and shoulders, of one month's duration. However, the present illness really began five years previously with a burning sensation behind the sternum. This occurred only on exertion. Electrocardiograms and x-rays of the chest taken then, were said to have been negative. He continued at work for a year although the sensation recurred. At the end of a year, another x-ray was made and he was told that his heart was enlarged. Aspirin and bicarbonate of soda appeared to afford some relief. He was sent to Arizona for six months but returned to New York unimproved. For

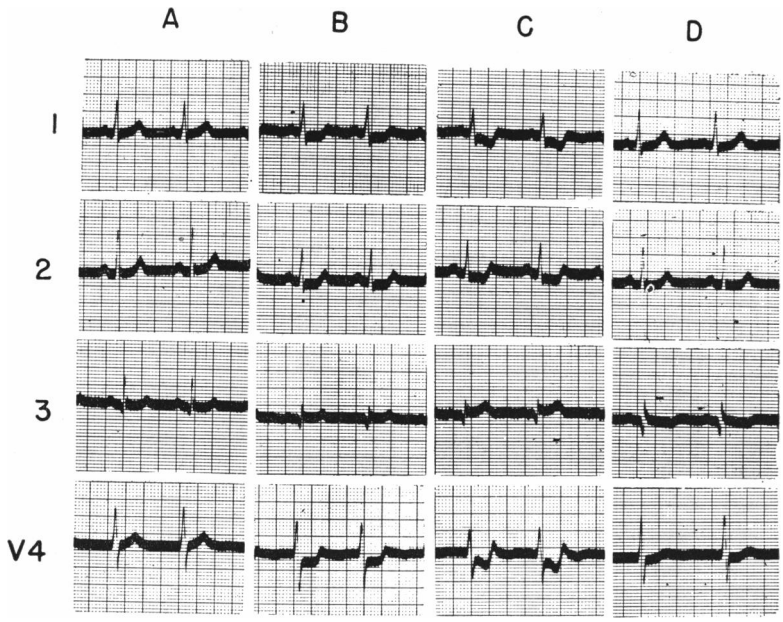

Fig. 2—Anoxemia test. A—control. B—after breathing 10 per cent oxygen for 5 minutes. C—after 20 minutes of hypoxia. D—5 minutes after breathing 100 per cent oxygen for 1 minute.

the two months prior to admission he complained of attacks of knife-like pain in the right shoulder which sometimes radiated to the right ring finger. These occurred chiefly in the evening before retiring. X-rays of the gallbladder revealed no stones and no disturbance in function. He stated that nitroglycerin relieved the pain in his right shoulder but did not affect the discomfort in his chest. His story rambled and was confusing.

Examination showed no cardiac enlargement on physical examination or by x-ray. There was a short, blowing systolic murmur at the apex. The blood pressure was 135 mm. Hg systolic; 90 diastolic. The blood count was normal. The venous pressure was 78 mm. of water. The circulation time, by the decholin method, was 20 seconds. X-rays of the gastrointestinal tract revealed no abnormalities. There were osteoarthritic changes in the thoracic spine, especially on the right side. An electrocardiogram, including precordial leads, was normal.

The anoxemia test was strikingly positive, as shown in Figure 2. There were changes in both the RS-T segments and in the T-waves.

The patient did not complain of discomfort during the test but at its conclusion said that after 10 minutes of hypoxia he experienced pain in the upper back on the right side and across the right chest. The oxygen saturation of the blood was determined during the period of hypoxia by the Millikan oximeter. The control reading was 97 per cent; after 5 minutes, 78 per cent; after 20 minutes, 75 per cent; 5 minutes after breathing 100 per cent oxygen for 1 minute, 98 per cent.

Recent observations by Gofman and his associates[19] indicate that variations in the patterns of lipoprotein in the blood serum, as studied with the ultracentrifuge, may reflect varying degrees of error in lipid metabolism. A strong positive correlation has been demonstrated between elevation of the larger "S_f 10-30" class of lipoproteins and the rate of development of atherosclerosis. Chemical changes in the blood and tissues may well furnish clues relating to the origin of arterial degeneration and it seems possible that such changes may be employed diagnostically to detect the basic metabolic fault in its early stages at a time when it can be retarded or reversed.

Summary

The existence of lesions in the coronary arteries has been known for several centuries but the correlation between the pathologic changes in these vessels and the clinical manifestations caused by them were slow to be recognized. It is only in the past thirty-five years that the medical profession has become familiar with the problems of coronary heart disease and aware of their importance. There has been an increase in the incidence of the diagnoses of coronary disease during this period, both at the bedside and at the autopsy table, as indicated by a survey of the records at the Columbia-Presbyterian Medical Center from 1910 to 1950. The upward trend in the frequency of clinical diagnosis has been greater than that in the autopsy series, probably due in large part to the development of greater diagnostic skill; however, there appears to have been a significant increase as found at autopsy, most marked during the decade 1910 to 1920.

The clinical manifestations of coronary heart disease are due to a functional disorder which, in all instances, is coronary insufficiency. A schema has been presented on the basis of which these features, acute or chronic, with or without infarction, may be viewed as effects of a disproportion between the amount of blood available for the myo-

cardium and the work which the heart is called upon to perform.

Certain diagnostic aids have been discussed and their value has been appraised. The solution to the problem of coronary heart disease is to be found in discovering the cause of atherosclerosis and eventual understanding of its nature will point the way to more effective therapy and, of even greater importance, to measures for prevention.

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